Abstract

C-reactive protein, cardiovascular diseases and endothelial dysfunction

Diploma thesis

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Background: Healthy endothelium significantly affects the vascular homeostasis which is damaged during the endothelial dysfunction. This dysfunction is linked to number of cardiovascular diseases. Endothelial dysfunction, together with the chronic inflammation, represents also basis of the metabolic syndrome. The aim of the thesis was to detect selected markers of endothelial dysfunction in recently published transgenic rat model of metabolic syndrome.

Methods: In this thesis, following factors were evaluated: endothelial nitric oxide synthase, phosphorylated endothelial nitric oxide synthase, superoxide dismutase 3, cyclooxygenase 2, nuclear factor κB and forkhead box P3. Evaluation of their expression in the aorta homogenates was performed by method Western blot. The control group was composed of spontaneously hypertensive male rats, the evaluated group was made from transgenic spontaneously hypertensive male rats expressing human C-reactive protein. Then the detection of proteins was performed by chemiluminiscent method on X-ray films. All the data were evaluated by densitometry method.

Results and conclusions: Our results show no significant changes in the expression of evaluated markers. The only one difference occured in superoxide dismutase 3 levels, there was more than double reduction of expression (40,92%). Decreased expression of superoxide dismutase 3 can indicate oxidative stress in transgenic rats expressing C-reactive protein, however, marked endothelial dysfunction wasn´t detected in this study.

Key words: C-reactive protein, endothelial dysfunction, metabolic syndrome, SHR-rats, nitric oxide, cyclooxygenase 2, superoxide dismutase 3, nuclear factor κB, FOXP3, Western blot.